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DNA discovery reverses wrinkles and hair loss

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New experiments with mice show that by treating a mutation-based imbalance in mitochondrial function, animals that looked physically aged regrew hair and lost their wrinkles restoring them to a healthy, youthful appearance in just weeks.

"To our knowledge, this observation is unprecedented," says geneticist Keshav Singh from the University of Alabama at Birmingham.

One of the focal points of anti-ageing research is investigating the so-called mitochondrial theory of ageing, which posits that mutations in the DNA of our mitochondria – the 'powerhouse of the cell' – contribute over time to defects in these organelles, giving rise to ageing itself, associated chronic diseases, and other human pathologies.

To investigate these mechanisms, Singh and fellow researchers genetically modified mice to have depleted mitochondrial DNA (mtDNA).

They did this by adding the antibiotic doxycycline to the food and drinking water of transgenic mice. This turned on a mutation which causes mitochondrial dysfunction and depletes their healthy levels of mtDNA.

In the space of eight weeks, the previously healthy mice developed numerous physical changes reminiscent of natural ageing: greying and significantly thinning hair, wrinkled skin, along with slowed movements and lethargy.

The depleted mice also showed an increased numbers of skin cells, contributing to an abnormal thickening of the outer layer of their skin, in addition to dysfunctional hair follicles, and an imbalance between enzymes and inhibitors that usually prevents collagen fibres from wrinkling skin.

But once the doxycycline was no longer fed to the animals, and their mitochondria could get back to doing what they do best, the mice regained their healthy, youthful appearance within just four weeks.

Effectively, they reverted to the animals they were before their mitochondrial DNA content was tampered with – which could mean mitochondria are reversible regulators of skin ageing and hair loss.

"It suggests that epigenetic mechanisms underlying mitochondria-to-nucleus cross-talk must play an important role in the restoration of normal skin and hair phenotype," says Singh.

"This mouse model should provide an unprecedented opportunity for the development of preventative and therapeutic drug development strategies to augment the mitochondrial functions for the treatment of ageing-associated skin and hair pathology and other human diseases in which mitochondrial dysfunction plays a significant role." Researchers said.